Covid research & implications

Purpose: This document is a running curation of Covid research cited in scholarly journals and/or trustworthy popular media, with the goal of forming a story about the implications of this growing data set on personal and public health.

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Study	Published	Main takeaways	Notes
Accelerated aging			
Evidence for Biological Age Acceleration and Telomere Shortening in COVID-19 Survivors	International Journal of Molecular Sciences	Covid infection shortens the telomeres and ages the body by 5 years.	“The results show a consistent biological age increase in the post-COVID-19 population, determining a DeltaAge acceleration of 10.45 ± 7.29 years (+5.25 years above the range of normality).”
Shorter telomere lengths in patients with severe COVID-19 disease	Aging	Covid infection shortens the telomeres, which ultimately shortens lifespan.	“Critically short telomeres impair the regenerative capacity of tissues and trigger loss of tissue homeostasis and disease.”
Accelerated	Nature	DNA analysis	“We also find the

biological aging in COVID-19 patients	Communications	shows people are biologically older than their age post Covid infection, regardless of previous health or severity of acute illness from Sars2 infection.	increasing acceleration of epigenetic aging and telomere attrition in the sequential blood samples from healthy individuals and infected patients developing non-severe and severe COVID-19”
Immune system impairment			
Reduction and Functional Exhaustion of T Cells in Patients With Coronavirus Disease 2019 (COVID-19)	Frontiers in Immunology	Covid infection is destroying and/or exhausting T cells which are a critical part of our immune system.	
AIDS and COVID-19 are two diseases separated by a common lymphocytopenia	Curated at Researchgate	Covid is more like airborne AIDS than the flu in terms of its impact on our immune system. In fact, it’s worse on our T cells.	“We observe a comparable reduction in B cells in both diseases and a more severe reduction in the total amount of T cells in COVID-19 as compared to AIDS patients”
Single-cell multiomics revealed the dynamics of antigen presentation, immune response and T cell activation in the COVID-19 positive and recovered individuals	Frontiers in Immunology	Covid infection is destroying naive T cells, suppressing B cell response, and triggering antigenic response (i.e. autoimmune response) which all serve to screw up an otherwise healthy immune system. This immune system damage is	“Importantly, we discovered that a subset of the naive T-cells from the healthy individuals ***were absent*** from the recovered individuals, suggesting a post-infection inflammatory stage.” (Emphasis added.)

		happening *after* acute infection, suggesting the body's immune system is struggling to cope with Covid long after someone "recovers" from it.	
SARS-CoV-2 Uses CD4 to Infect T Helper Lymphocytes	Curated at Medrixiv (pre-print)	Covid directly infects the cells, persists in bodily reservoirs, and uses the CD4 pathway to infect our lymphocytes, dysregulating our immune system.	"SARS-CoV-2 infected T helper cells express higher amounts of IL-10, which is associated with viral persistence and disease severity. Thus, CD4-mediated SARS-CoV-2 infection of T helper cells may explain the poor adaptive immune response of many COVID-19 patients."
Superantigenic character of an insert unique to SARS-CoV-2 spike supported by skewed TCR repertoire in patients with hyperinflammation	Proceedings of the National Academy of Sciences (PNAS)	Covid is hyperactivating the body's immune response in a similar way to bacterial superantigens, leading to things like MIS-C, cytokine storms, and toxic shock syndrome.	"Notably, the superantigen-like motif is not present in other SARS family coronaviruses, which may explain the unique potential for SARS-CoV-2 to cause both MIS-C and the cytokine storm observed in adult COVID-19."
SARS-CoV-2 infection causes immunodeficiency in recovered patients by downregulating	Nature	Covid infection causes lasting destruction to our immune system.	"We found that SARS-CoV-2 infection causes immunodeficiency in recovered patients"

CD19 expression in B cells via enhancing B-cell metabolism			
Immunological dysfunction persists for 8 months following initial mild-to-moderate SARS-CoV-2 infection	Nature	“Long Covid” is likely one manifestation of a crushed immune system from Covid infection. (Note that this study does not suggest things get better after 8 months - that’s just the time horizon for the study.)	“Patients with LC had highly activated innate immune cells, lacked naive T and B cells and showed elevated expression of type I IFN (IFN-β) and type III IFN (IFN-λ1) that remained persistently high at 8 months after infection.”
Transcriptional reprogramming from innate immune functions to a pro-thrombotic signature by monocytes in COVID-19	Nature	Covid-19 is messing with our monocytes, which are key white blood cells in our immune system, specifically reprogramming them to be “functionally pro-thrombotic” or prone to triggering blood clotting. This is a really scary finding.	“We find that ex vivo isolated CD14+ monocytes from mild and moderate COVID-19 patients are phenotypically different from monocytes from healthy individuals.”
Brain damage			
Is the Frontal Lobe the Primary Target of SARS-CoV-2?	Journal of Alzheimer’s Disease	There’s growing evidence that Covid infection attacks the brain’s frontal lobe, which is responsible for higher executive functions, which could explain why so many neuropsychiatric symptoms are	“...[T]here is some evidence that SARS-CoV-2 could preferentially and directly target the frontal lobes, as suggested by behavioral and dysexecutive symptoms, fronto-temporal hypoperfusion on

		reported in connection with Covid.	MRI, EEG slowing in frontal regions, and frontal hypometabolism on 18F-FDG-PET imaging.”
SARS-CoV-2 drives NLRP3 inflammasome activation in human microglia through spike protein	Molecular Psychiatry (scholarly journal) Iflscience (reputable science news site)	Covid infection is causing the same inflammation that leads to Parkinson’s, Alzheimer’s and other forms of dementia.	Builds on a growing body of research that Covid directly interacts with microglia and triggers inflammatory response in the brain.
SARS-CoV-2 is associated with changes in brain structure in UK Biobank	Nature (scholarly journal) NY Times	Infection strongly correlated with (1) reduction in grey matter in the brain (2) brain tissue damage (3) reduction in overall brain size	UK-based study by a dozen+ researchers used MRI scans and controls for pre-existing conditions.
SARS-CoV-2 promotes microglial synapse elimination in human brain organoids	Molecular Psychiatry	Covid infection is likely destroying physical synapses in the brain.	This is an extremely worrisome impact of post-acute Covid.
MRI Reveals Significant Brain Abnormalities Post-COVID	Imaging Technology News (reporting on data from a study that was published in late 2022.)	“MRI results showed that patients who recovered from COVID-19 had significantly higher susceptibility values in the frontal lobe and brain stem compared to healthy controls. The clusters obtained in the frontal lobe primarily show	“These brain regions are linked with fatigue, insomnia, anxiety, depression, headaches and cognitive problems,” Mishra said. It’s especially concerning that Covid infection seems to target the frontal lobe, where higher

		differences in the white matter.”	executive functions take place.
Stroke			
COVID-19 related stroke in young individuals	The Lancet Neurology	Covid causes clotting and increases the risk of stroke exponentially for people under age 50.	“[D]ata supporting an association between COVID-19 and stroke in young populations without typical vascular risk factors, at times with only mild respiratory symptoms, are increasing.”
Acute Ischemic Stroke During the Convalescent Phase of Asymptomatic COVID-2019 Infection in Men	JAMA	Covid infection doubles the risk of stroke in men under 50, with the average onset of stroke two months post-infection.	“Acute ischemic stroke could be part of the next wave of complications of COVID-19, and stroke units should be on alert and use serological testing, especially in younger patients or in the absence of traditional risk factors.”
COVID-19 diagnosis raises risk of heart attack, stroke	Curated in Harvard Health Publishing Research originally published in The Lancet	Risk of stroke and heart attack increases exponentially in the weeks and months after Covid infection.	“COVID-19 infections appear to be especially risky, perhaps because they trigger an exaggerated inflammatory response that makes blood clots more likely.”
Risks of deep vein thrombosis, pulmonary	BMJ	People with Covid have a significantly	“Compared with the control period, incidence rate

embolism, and bleeding after covid-19: nationwide self-controlled cases series and matched cohort study		elevated risk of blood clots, stroke, and bleeding for a long time after acute illness is over.	ratios were significantly increased 70 days after covid-19 for deep vein thrombosis, 110 days for pulmonary embolism, and 60 days for bleeding.”
Heart attack / heart disease			
Excess risk for acute myocardial infarction mortality during the COVID-19 pandemic	Journal of Medical Virology	Before the pandemic, death by heart attack was dwindling. It has skyrocketed amid Covid, with younger people dying of heart attacks. Strong correlation to Covid infection.	“The trend of mortality suggests that age and sex disparities have persisted even through the recent Omicron surge, with excess AMI-associated mortality being most pronounced in younger-aged adults.”
COVID and the Heart: It Spares No One	Johns Hopkins	Covid infection does tremendous damage to the endothelial layer of our blood vessels and reprograms some white blood cells to induce clotting instead of fighting pathogens. These problems persist well beyond the acute illness period and may cause lasting damage.	“[P]eople with COVID-19 have a higher risk of all sorts of heart problems at one year. That included arrhythmias (irregular heart beats or the heart beating too fast or too slow) and atrial fibrillation (a fast heart rhythm in a particular pattern). We found evidence of an increased risk of stroke, of blood clots in the legs and the lungs, and of heart failure and heart attacks.”

Impact on kids			
Covid-19 is a leading cause of death in children and young people ages 0-19 years in the United States	Curated in medrixv	Contrary to popular narratives, Covid has been a leading cause of death for young people since the pandemic began, and it was especially bad between the springs of 2021 and 2022.	“Due to the impact of mitigations such as social distancing and our comparison of a single disease (Covid-19) to groups of causes such as deaths from pneumonia and influenza, these rankings are likely conservative lower bounds.”
Post-COVID-19-associated morbidity in children, adolescents, and adults	PLOS Medicine	Covid significantly increases the chance of death in children, adolescents, and young adults across multiple risk areas.	“We observed significant new onset morbidity in children, adolescents, and adults across 13 prespecified diagnosis/symptom complexes, following COVID-19 infection.”
On the role of different age groups in propagating Omicron epidemics in France	BMJ	Kids *do* spread Covid, and in fact were the largest vectors of spread during the Omicron wave in early 2022.	“Children aged 10-19y played the greatest relative role in propagating Omicron epidemics, particularly when schools were open, followed by children aged 0-9y and adults aged 20-29y, as well as adults aged 30-49y.”
Delta and Omicron killed far more children than flu ever does.	Inside Medicine	Covid is killing kids at 6x the rate of seasonal flu.	“Make no mistake: Covid-19 is imparting an unusual burden on children compared to all other

			respiratory viruses.”
Multisystem inflammatory syndrome in children (MIS-C) showing disseminated aspergillosis, cytomegalovirus reactivation and persistent SARS-COV-2: Case report with autopsy review	Pathology Research and Practice	Some kids, probably about 1%, who are infected with Covid experience severe autoimmune responses like MISC-C and “cytokine storm,” which can be fatal.	“Although MIS-C is generally considered to be a post-infectious hyperimmune reaction, persistence of SARS-COV-2 is a feature in all autopsies of MIS-C patients reported to date, suggesting a possible role in the pathogenesis, at least in fatal cases.”
Diabetes			
Diabetes risk rises after COVID, massive study finds	Nature News (but the study was published in The Lancet Diabetes & Endocrinology)	Huge study, 180k people, showed that risk of new onset diabetes goes up significantly after even mild Covid infection, with more severe acute disease increasing the odds of diabetes considerably.	These same researchers used a similar methodology to show strong connection between Covid infection and kidney disease, heart failure, and stroke.
Association of COVID-19 with diabetes: a systematic review and meta-analysis	Nature	This meta analysis looked at eight studies and found that Covid infection has a strong correlation to new onset diabetes.	“In this systematic review and meta-analysis, COVID-19 was associated with higher risk for developing new onset diabetes among survivors. Active monitoring of glucose dysregulation after recovery from severe acute respiratory

			syndrome coronavirus 2 (SARS-CoV-2) infection is warranted.”
Sexual health			
Investigation of the effect of COVID-19 on sperm count, motility, and morphology	Journal of Medical Virology	Men with Covid have reduced sperm count, suggesting the virus and subsequent disease impact male reproductive health.	“It was observed that men with COVID-19 had decreased sperm concentrations suggesting that COVID-19 may have a negative effect on male fertility.”
Tip of the iceberg: erectile dysfunction and COVID-19	Nature	Covid is likely contributing to male erectile dysfunction by destroying the endothelial layer of key blood vessels (which is a known Covid impact throughout the body) and directly damaging the testicles.	“In summary, COVID-19 infection could affect male sexual function through endothelial damage in erectile tissue, testicular damage, and psychological alterations.”
Pregnancy & fetus development			
Comparison of Pregnancy and Birth Outcomes Before vs During the COVID-19 Pandemic	JAMA	Covid-19 is associated with increases in pregnancy-related complications and maternal deaths during delivery hospitalization.	“While live-birth outcomes and mode of delivery remained stable, small but significant increases in pregnancy-related complications and maternal death during

			delivery hospitalization were observed.”
SARS CoV-2 infection as a risk factor of preeclampsia and pre-term birth. An interplay between viral infection, pregnancy-specific immune shift and endothelial dysfunction may lead to negative pregnancy outcomes	Annals of Medicine	SARS-CoV-2 having affinity to ACE-2 and causing it’s downregulatio n receptor may cause endothelial injury leading to compliment activation and formation of NETs, together with RAS dysregulation this may cause preeclampsia to develop in pregnant patients.	PTB may occur in patients as an effect of SARS-CoV-2 infection in first or second trimester as an effect of TLR4 pathway dysregulation with lower levels of IFNβ.
Sex-Specific Neurodevelopment al Outcomes Among Offspring of Mothers With SARS-CoV-2 Infection During Pregnancy	JAMA	These findings suggest that male offspring exposed to SARS-CoV-2 in utero may be at increased risk for neurodevelopme ntal disorders.	This cohort study of 18 355 infants delivered after February 2020 found that male but not female offspring born to mothers with a positive SARS-CoV-2 polymerase chain reaction test result during

			pregnancy were more likely to receive a neurodevelopmental diagnosis in the first 12 months after delivery, even after accounting for preterm delivery.